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January 1953

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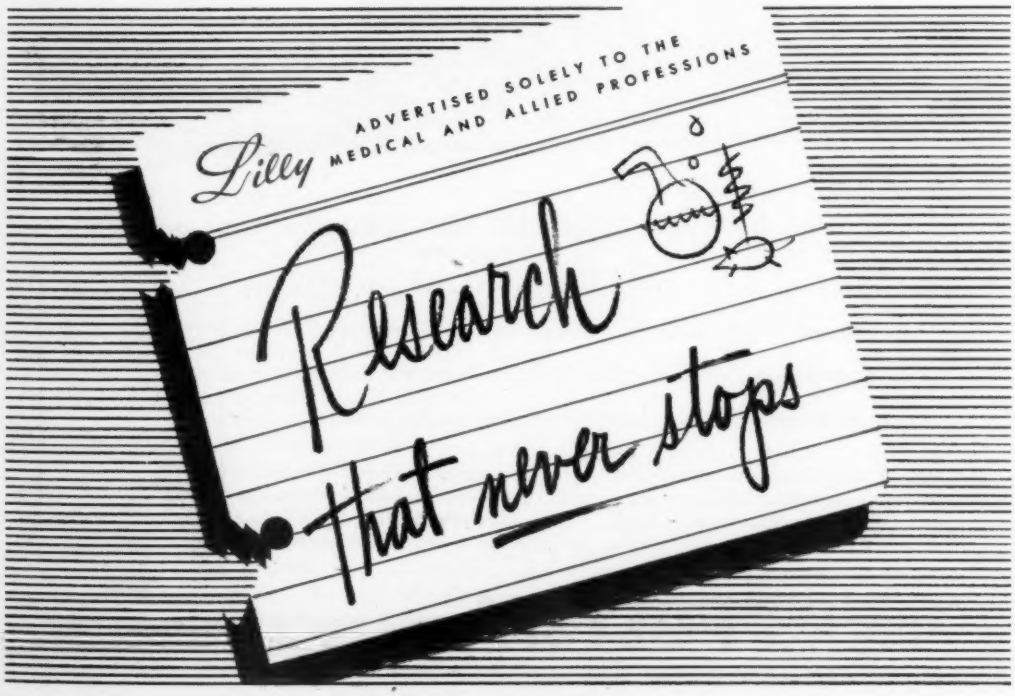

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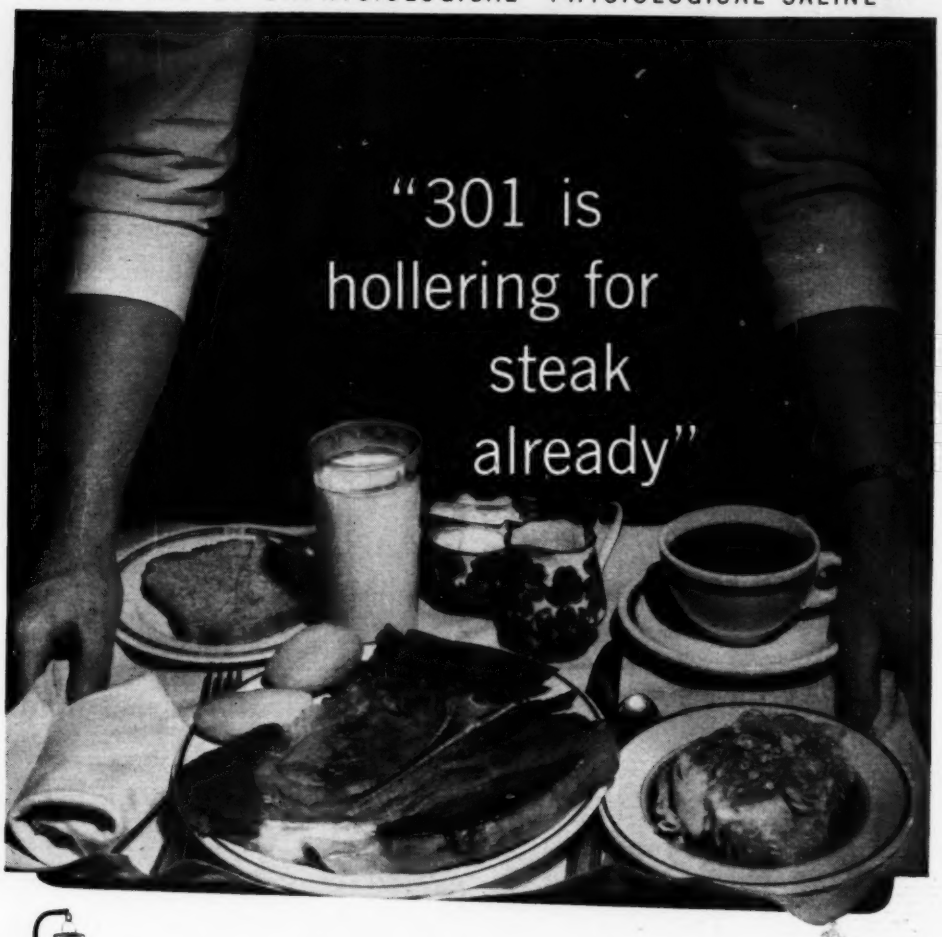

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1. Fox, C. L. Jr., et. al.: An Electrolyte Solution Approximating Plasma Concentrations with Increased Potassium for Routine Fluid and Electrolyte Replacement. J. A. M. A., March 8, 1952.

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De Rebus Medicis Et Politicis

BY ROBERT B. HOMAN, JR., M. D., EL PASO, TEXAS
MEMBER OF THE HOUSE OF DELEGATES OF THE AMERICAN MEDICAL ASSOCIATION

A FOUR YEAR TRIAL

It appears that we have a man of action entering the White House on January 20, 1953. This is written as General Eisenhower is returning from Korea — the end of a trip promised the American people during the campaign. As this is written, the President-Elect has not yet met with General MacArthur, and his decision on the Korean War has not been announced. But, as of today, he has certainly plowed a deep and straight furrow toward a dynamic administration, of which he has established himself as the absolute boss.

The unprecedented manner in which the General appointed his cabinet and his advisors far in advance of his inauguration is evidence of his desire for quick action. In the selection of this cabinet and of his assistants, he has indicated that he has taken his election to mean that Americans really want a change. The egg-heads are leaving Washington and business men are moving in. The General's famed organizational ability has been well illustrated since November fourth!

It definitely appears that freedom of opportunity — free enterprise — is going to have another chance in this country. Thereby hangs the theme of this article.

ON TRIAL

Several days ago one of my good doctor friends remarked that the medical profession of this country is now "on trial" for four years! Nothing could be more true. During the past few years the profession has been fighting new and fair deal socialistic tendencies and praising the system of free enterprise not only in medicine but in business in general. The fight has thus far been successful, but there are many medical problems yet to be solved to the satisfaction of the American people. It behooves the profession to further examine these problems, and make some progress toward their solution or lose our case forever.

At various times this column has discussed some of these matters of medical importance. Of major concern to the people is an adequate distribution of doctors and medical facilities in rural areas. This naturally brings into consideration the problems of medical education and over-specialization; the financing of our educational system; the actual "production" of more doctors, nurses, and technicians for an expanding population. These are not simple subjects to be easily conquered—they will require the cooperation of industry, labor, local and state governments, and the common citizens to bring about their solution. To maintain professional freedom in the process will be dificult indeed.

ECONOMIC PROBLEMS

The economic problems of medical care have not changed with the election. The cost of hospitalization continues to rise commensurate with higher and higher operating costs. The profession must continue to actively support the voluntary hospital and medical care insurance programs until every possible American family has adequate insurance coverage. And the doctors must continue to give their services even more abundantly to those in need of medical care.

Actually the entire industrial system of this country — the American Dream — is on trial for four years. Freedom of opportunity will exist in this country just so long as our system provides relative economic freedom to the vast majority of the people. The election of the new administration should provide an incentive to every freedom—loving American to produce and provide as never before.

OBSTETRICS

Post-Mortem Caesarean Section With Survival Of Twins

Teter, L. F., J. Michigan M. S. 51:1013, 1952

A lapse of six minutes is generally considered to be the maximum time that a fetus can survive *in utero* following the mother's death. In this case the elapsed time was ten to twelve minutes. It is stated that, legally, a doctor does not need the husband's or family's permission to perform a post-mortem section, although it is preferable to obtain permission if possible.

Lelia Y. Post Montgomery Hosp.

Clinical Clippings, December, 1952.

APHORISMS
TRUTHS AND CONCEPTS CONCERNING THE BRONCHOPULMONARY SYSTEM

By Andrew M. Babey, M. D., Las Cruces, N. M.

(CONTINUED)

(29)

"Pneumothorax treatment should not be given to patients with lung abcess except in rare instances as a life saving measure in hemorrhage." — J. B. Amberson, Jr., *loc. cit.*

(30)

"Every case of lung abscess should be bronchoscoped." — J. B. Amberson, Jr., *loc. cit.*

(31)

"It is not uncommon for a pluerisy with effusion to start exactly like a pneumonia with a chill, fever (up to 105) and stitch in the side." — J. B. Amberson, Jr., *loc. cit.*

(32)

"Pleural crepitations heard above the level of a resolving pleural effusion may at times deceive you into thinking you have parenchymal involvement." — J. B. Amberson, Jr., *loc. cit.*

(33)

"Recurrent unilateral or bilateral spontaneous pneumothorax is very commonly due to rupture of superficial emphysematous blebs." — J. B. Amberson, Jr., *loc. cit.*

(34)

"If you get a musty or odorous pus and a culture is negative, don't be deceived. Do an anerobic culture." — J. B. Amberson, Jr., *loc. cit.*

(35)

"If you find lung abscess in a man over 50, think of carcinoma of the bronchus as the cause." — J. B. Amberson, Jr., *loc. cit.*

(36)

"Bronchiectesis is almost never diagnosed by bronchoscopy." — J. B. Amberson, Jr., *loc. cit.*

(37)

"Congenital cystic disease of the lungs is more mystic than cystic." — (Quoted by J. B. Amberson, Jr., after Grethmann), *loc. cit.*

(38)

"The more fibrotic and emphysematous a lung, the more atypical will the pneumonia be." — J. B. Amberson, Jr., *loc. cit.*

(39)

"Every chest infection damages the lungs." — J. B. Amberson, Jr., *loc. cit.*

(40)

"Before operating on anyone with much laryngeal involvement, take a chest plate to be sure there is no aspiration pneumonia." — J. B. Amberson, Jr., *loc. cit.*

(41)

"Fungus disease of the lung usually turns out to be tuberculosis, and frequently atypical hematogenous tuberculosis, for fungus disease is uncommon." — J. B. Amberson, Jr., *loc. cit.*

(42)

"In any pyrexia of long duration, look closely at the mediastinum for tuberculous glands." — J. B. Amberson, Jr., *loc. cit.*

(43)

"An apical pneumonia is likely to be missed because of the predominance of cerebral symptoms." — T. Grier Miller, *Medical Lectures,* 1922.

(44)

"When acute disease of the lungs occurs in persons with rickety deformity, the violence of the symptoms is often quite disproportionate to the extent and severity of the pulmonary disease, and may thereby suggest unnecessarily active treatment." — Thomas Addison's Works, *New Syndenham Soc.,* London, 1868 vol. 36, page 65.

(45)

"This pungent (heat of the skin) though not necessarily present is very rarely absent so long as any portion of the lung continues to be in the first or crepitating stage of pneumonia." — Thomas Addison's Works, *loc. cit.*

(46)

"When the anterior and inferior portion of the left lung is consolidated by pneumonia, percussion may produce good resonance in consequence of the flatulent stomach and thereby throw us off our guard. When pneu-

monic consolidation takes place anteriorly and inferiorly, and even posteriorly on the right side a remarkable degree of resonance is occasionally elicited in the highly tympanitic condition of the intestines. Under the same conditions auscultation may detect a well-marked modification of amphoric respiration, and metallic tinkling, to a considerable height in the chest thereby leading to the erroneous conclusion that pneumothorax is present." — Thomas Addison's Works, *loc. cit.*

(47)

"We may be called to see a case of pleurisy before a single physical sign has developed. In such a case it may be doubtful whether the pain arises from pleurisy, rheumatism, neuralgia, or the approach of shingles." — Thomas Addison's Works, *loc. cit.*

(48)

"When the effusion into the chest is of the purely serous kind, or when the proportion of albuminous material is very inconsiderable, the fluid gravitates to the floor of the cavity and may, unless very abundant, entirely escape detection either by auscultation or percussion." — Thomas Addison's Works, *loc. cit.*

(49)

"Sound is transmitted through bodies by a successive motion of its molecules, the whole mass not necessarily undergoing any change of place, whilst the movement appreciable by the hand is the change of place which an entire body undergoes." — Thomas Addison's Works, *loc. cit.*

(50)

"I shall content myself with offering a caution not to mistake the crepitations so often attendant upon recent pleuritic effusion for the crepitatory and mucous rales of bronchopneumonia." — Thomas Addison's Works, *loc. cit.*

(51)

"The reason that most metastatic pulmonary malignancies are shown as rounded, sharply defined opaque shadows in the roentgenogram is due to the large number of end arteries in the lungs and to the yielding nature of lung parenchyma. A malignant cell lodges in an end artery and multiplies; as the resistance in all direction is the same it grows as a round, discrete tumour." — Radiologic Maxims, *Mississippi Valley Med. Jour.*, 61: 114, 1939.

New Interns Appointed By National Matching Plan

A national matching plan has been used for the first time this year to place new interns in hospital appointments.

The plan was designed to help lessen the confusion which has occurred in the past when hospitals were seeking 10,000 interns from a graduating class of 6,000.

Under the plan sponsored by the National Interassociation Committee on Internships, hospitals and students contact each other freely during the student's senior year.

Students apply for any internship which interests them and visit hospitals of their choice. After these preliminaries have been completed, students and hospitals file confidential ratings with the Committee.

The two are then matched, with the student receiving the internship he prefers most, if this agrees with the hospital's rating of him.

Success of the plan is indicated by the fact that 84 per cent of this year's new interns were matched with the hospital they indicated as their first choice. An additional 10 per cent received their second choice placement. Seventy-four per cent of the hospitals received the student they designated as their first choice.

From Medical Grand Rounds*

These notes are abstracts of opinions expressed by staff members during case presentations at the Medical Grand Rounds of the Pratt Diagnostic Clinic, Boston.

▼ ▼ ▼

The possibility of recurrent attacks of acute infectious mononucleosis is very rare. So-called "chronic infectious mononucleosis" has been reported but it is doubtful that there is such an entity. It may have been, and probably was, a coincidence that Hodgkin's disease developed in a patient who had recently had definitely proved infectious mononucleosis. It has, however, been postulated that Hodgkin's disease may represent neoplastic or granulomatous response in lymph nodes to previous infection with tuberculosis, brucellosis, or chronic sepsis, and this case raises the possibility that infectious mononucleosis might also animate a similar chain of events. The possibility is remote, however, because the association between infectious mononucleosis and Hodgkin's disease has not previously been noted although both of these diseases are fairly common.

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SURGICAL MANAGEMENT OF HYPERTENSION

By W. A. Jones, M. D., El Paso

At the present time all that we can offer a patient who is suffering from hypertension is palliative measures such as medical management by specific diets and surgery on the autonomic nervous system. First it might be interesting for a few moments to go into the history of surgery of the autonomic nervous system. It was first considered in 1923 by Danielopulo. In 1927 Pieri described an anatomical approach for splanchnic surgery; in 1929 he resected the splanchnic nerve for atony of the stomach. He performed this operation on two or three occasions and noted a lowering of the blood pressure. In May 1930, he performed a splanchnicectomy for hypertension. Although the results were somewhat encouraging we have no record of this procedure being applied again until around about 1933 when Peet started what is now called a Peet operation for hypertension. Crile in 1936 developed what he called a celiac ganglionectomy and used this procedure on several occasions with generally unsatisfactory results. The other popular operative procedure at this time is known as the Smithwick operation for hypertension.

NO DEFINITE RULE

In the selection of cases it might be stated that no definite rule can be set down, but in general we would like to have the patient under 50 years of age; if he is over 50 and he has evidence of severe headaches and failing of vision, vertigo, etc., exception to the rule is made. A complete work-up should be made from the cardiac stand-point, including electro-cardiogram and X-ray of the heart. One should look for edema of the extremities, anginal pain, shortness of breath, or any findings that would suggest cardiac decompensation. If the patient should show any evidence of cardiac decompensation surgery is contra-indicated at that time. However, if he becomes compensated after a reasonable length of time under medical management, surgery should then be considered. A recent coronary occlusion is also a contra-indication for surgery. The patient should be checked from a genito-urinary standpoint, for blood urea nitrogen, concentration test, urea clearance, P. S. P. and I. V. pyelogram and retrograde pyelogram if indicated. Chronic glomerulonephritis is a contra-indication for surgery. If urea nitrogen is above 45 it is usually associated with severe renal impairment and this too is a contra-indication for surgery.

CEREBRAL ACCIDENTS

Cerebral vascular accidents, if minor in type are not a contra-indication; but if a patient has a hemiplegia or aphasia or other evidence of cerebrovascular disease, the operation is not justified.

A sleep test with three grains of sodium amytal is given at hourly intervals and the blood pressure taken every 30 minutes, and if you get adequate drop in systolic and diastolic pressure readings, this is sometimes a good indication as to the results that you will obtain, but not necessarily so.

According to Keith, Wagner and Barker of the Mayo Clinic, a prognosis can be determined fairly accurately according to the eye findings.

Group 1: Mild narrowing of and sclerosis of the retina vessels.

Group 2: Moderate to marked sclerosis of the retinal vessels characterized by exaggeration of the arterial reflex and arterio-venous compression.

Group 3: Patients have angio-spastic retinitis characterized by edema, cottonwool exudate, hemorrhages in the retina superimposed on the sclerotic spastic lesions of the arteries.

Group 4: If there is measurable edema of the optic disc plus the findings in Group 3 comprise Group 4.

In comparison of medical management of patients according to the above classification and to surgical management, there is no doubt that the life of the patient has been prolonged by surgical intervention. According to Smithwick non-surgical series of patients the average mortality rate followed from four to 10 years classified according to his groups of one to four, the over-all mortality rate was 51 per cent. Hammarstrom and Bechgaard in 1950 followed 435 cases from two to 10 years, mortality rate was 51 per cent. Smithwick's surgically treated patients, of 596 cases, the over-all mortality rate followed from four to 12 years was 30 per cent. Series

of surgical procedure tabulated by various operators from years 42 to 48 is as follows:

> Peet in 1948 reported in 2000 cases, average age 40, followed from one to 15 years, mortality rate 1.5 per cent, surgically; he had lowering of blood pressure in 81.3 per cent and symptomatic relief 86 per cent. Various authors reported symptomatic relief from 80 to 90 per cent in surgical procedures.

TYPE OF OPERATION

As to the type of operation I think it probably varies with the operative technique which one has been trained to do. Peet's operation is removal of the splanchnic nerves and a dorsal ganglionectomy from about D-6 to about D-12 bilaterally whereas Smithwick is from about D-6 down to and including L-1 and L-2.

CONCLUSION

1) There is no doubt that surgery has a definite place in the treatment of hypertension.
2) The best results are obtained from young patients before there is gross damage to the extra renal vascular bed.
3) It is believed that surgery should be adequate, which calls for bilateral removal of the splanchnic nerves and a dorsal ganglionectomy from D-4 down to L-2 bilaterally.
4) The majority of far advanced cases we can only hope for symptomatic relief but ofttime the results are quite gratifying and the patient is made relatively comfortable, this justifies the operative procedure. As stated in the beginning the selection of cases is an individual one but if there is evidence of marked renal involvement surgery is contra-indicated.

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New Desserts Remove Excess Salt

At the recent convention of the American Medical Association, visiting physicians made thousands of taste tests of cookies into which there had been baked a mixture of unflavored and finely ground sodium-removing resins. The doctors approved the medicated cookies for taste; many believed they compared favorably with grandma's best.

Cookies are but one foodstuff into which the housewife may blend this new form of an already established agent which removes excess sodium from the bodies of patients who suffer from heart disease, cirrhosis of the liver, edema of pregnancy, or hypertension. Fruit juices, milk, fudge, cake, and flavored gelatin are some of the other nutriments which will hold and disguise the new medication.

Now patients with excessive sodium retention can actually enjoy taking a medicine which allows other items in their diet to be seasoned more liberally and tastefully with once-forbidden salt. Eli Lilly and Company will supply physicians with books of various kitchen-tested recipes which may be given to patients.

Following its debut at the A.M.A. convention 'Carbo-Resin' (Sodium Removing Resins, Lilly), Unflavored, has been placed on the prescription market.

PEDIATRICS

Ineffectiveness Of Carob Flour In Infantile Diarrhea

Walker, S. H., A. M. Am. J. Dis. Child. 84:309, 1952

Several reports have recently appeared concerning the effectiveness of carob flour* in treatment of infantile diarrhea. From this critical study it is concluded that use of the flour does not reduce fluid loss or otherwise alter the course of the diarrhea. Carob-treated patients pass liquid stools throughout the usual course of the disorder. The formed mass seen in diapers of such patients is the result of consolidation of water by the hygroscopic flour and absorption of fluid by the diaper. This is not an indication of therapeutic effectiveness but may be of some practical value from a nursing standpoint.

**Arobon (Nestle Co.)*

Clinical Clippings, December, 1952.

RECURRENT, PAROXYSMAL AURICULAR TACHYCARDIA FOR TWENTY-FIVE YEARS; REPORT OF A CASE TREATED SUCCESSFULLY

By Andrew M. Babey, M. D., F. A. C. P., Las Cruces, New Mexico

Rapid, regular heart action occurring only occasionally, in paroxysms, is a commonplace in office and hospital practice, but when it becomes a chronic ailment appearing over two decades of a man's life it is worthy of note. The case we are reporting is just such a rarity, and since it illustrates most of the salient points of this distressing disorder we present it in some detail.

T. G., 69 year old white male, was seen for the first time February 3, 1951, being referred by Dr. Leland S. Evans of Las Cruces, New Mexico. His chief complaints were intermittent, rapid heart beat for twenty-five years, asthma and arthritis.

Twenty five years ago, while working in the Texas oil fields, the patient had his first bout of paroxysmal tachycardia during which his heart "snapped" suddenly and pounded very fast. He felt faint and weak. After some minutes the heart suddenly stopped racing, and except for fatigue he felt quite well. Thereafter, for the next twenty five years, these attacks recurred at least once a week. On one occasion the rate was said to be 165. After a year, the spells increased to several per day and he was advised to stop all heavy work if he wished to "stay alive."

BUYS FARM

The patient bought a farm, assigned all hard work to laborers and for the next five years was fairly comfortable. Bouts of tachycardia still occurred but were of short duration. One night, about five years after quitting the oil fields, a ditch on his farm suddenly gave way, and since no help was available, the patient shovelled dirt as fast as he could for about fifteen minutes to plug the gap. His heart then suddenly "snapped" and began to race so rapidly that he thought it "would bore a hole" in his chest. He barely managed to get home and fell into bed exhausted. About four hours later a doctor administered a hypodermic of morphine sulphate, and when he awakened from a long sleep the tachycardia had disappeared. He stayed in bed for about three days because of profound weakness, and was then told that he must give up his farm and "take things easy," whereupon he began to trade cattle. The attacks of rapid heart action still appeared about once a week despite this more leisurely life, and morphine injections were still his sole relief. After four years of this life, without known cause, he had a severe bout of tachycardia which would not stop despite several injections. Because of this resistant attack, he was admitted to the local hospital where he took oxygen for seven days, partly for the tachycardia and partly for the weakness which followed when it finally abruptly ceased. He was sent home and advised to rest in bed for one month. While in bed he continued to have bouts of tachycardia relieved by morphine injections.

The patient then returned to his work as a trader and got on fairly well except for having to see the doctor at almost regular seven day intervals for a hypodermic. It was his habit to go to the doctor's office, get his "shot," go home to bed, and after a long sleep awaken without tachycardia.

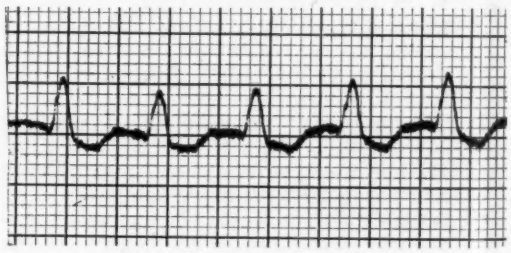

Figure 1. EKG taken during P. A. T. — rate 166.

ANOTHER BOUT

In 1946, again for no apparent reason, he had another prolonged bout of tachycardia lasting two days. He was taken to a hospital in El Paso where the rate was finally controlled by the administration of two ounces of ipecac which caused hours of vomiting. After a week's stay in the hospital he returned home and once more had to have morphine injections about once every seven days for his attacks of palpitation. In 1948 the patient was referred to a hospital in El Paso because of pneumonia during the course of which he suffered from frequent bouts of tachycardia. After returning home his trouble resumed its previous pattern.

Early in 1950 the patient again contracted

pneumonia and, while febrile, was prostrated by a two day bout of tachycardia for which he was transferred to El Paso. When he returned home his tachycardia became worse, the bouts lasting longer and coming more frequently, so that he was confined to bed the entire summer. Palpitation at this time was associated on five different occasions with severe substernal pain radiating into the left arm; the arm almost "broke off." By September 1950 it was plain that the patient had become addicted to morphine and dilaudid, the latter being prescribed since 1943 for chronic cough. Because of this complication he was again admitted to a hospital in El Paso where he stayed for four weeks. There he showed extreme withdrawal symptoms but finally overcame his difficulty and was allowed home, where he again began to suffer from paroxysmal rapid heart action, this time allowed to run its course as the patient refused to take any more "shots."

RHYTHMIC RECURRENCE

When the patient was first seen by us in February 1951 he was almost completely invalided by recurrent attacks of tachycardia. They happened at any time, when sitting, standing or lying down; when on a light or heavy diet; when smoking or not smoking; when irritated or placid. In fact, the patient had never been able to discover any provoking cause and was despondent about his future. At times he thought gas in the stomach produced the attacks, for he always felt bloated when the heart raced. However, all sorts of diets and intestinal medicines did not disturb the almost rhythmic recurrence of attacks. Of one thing he was certain — the attacks never occurred at night.

A review of the remainder of the patient's history revealed no recent weight loss, unusual sweating or tremor. He felt weak but mostly in the hands where rather marked fusiform joints were evident. In childhood he had "all the usual diseases." As an adult he had two bouts of pneumonia, and asthmatic bronchitis had been troublesome for at least seven years.

Previous Residence: Never outside the U.S.A.

Former Operations: In 1920 all his teeth were extracted because of pyorrhea.

Previous Injuries or Accidents: Broken forearm at the age of 10.

Marital History: Married 50 years, seven children; six living and well; wife living and well.

Habits: Poor sleeper, partly due to asthma and partly due to "nerves." Takes no alcohol; coffee and tea in moderation; no tobacco recently on advice of physician.

Family History: Mother died at 74, cause not known; Father died at 67, cause not known; two brothers, six sisters, all well except one brother who was killed in a car accident.

Inquiry by Systems:

Head: Rare headaches; no dizziness or trauma.

Eyes: The right eye was removed in 1915 following serious infection.

Ears: Hearing on right definitely impaired for an unknown period of years.

Nose, Mouth, Tongue, Throat, Neck: Non-contributory.

Respiratory: Chronic cough with abundant thick, grey sputum for about seven years; asthmatic attacks for same period with audible wheezing from time to time; no hemoptysis or prolonged fever.

Cardiac: Dyspnea occasionally on mild exertion with some wheezing; no foot or ankle edema; no orthopnoea. He has always slept on two pillows.

Gastro-intestinal: Hearty appetite; considerable gas and distress with or without palpitation; no jaundice, bloody or black stools; no hemorrhoids; no hematemesis. In 1949 a gastro-intestinal x-ray series revealed normal gallbladder, esophagus, stomach and duodenum. There were some diverticuli in the sigmoid colon. No paraesophagal hernia was reported.

Genito-urinary: Nocturia 2-3 times for several years.

Endocrine: Prefers warm weather.

Neuromuscular: More nervous recently; anxious and worried. For four months he has noticed considerable pain in the left shoulder when raising his arm upward or when putting on his coat.

Skin: Non-contributory.

Allergy: No hives or hay fever; no known food or pollen allergy. Dust storms provoke cough and wheezing.

Bones and Joints: The fingers of both hands have been stiff and swollen for about four months and he is unable to button his shirt without discomfort.

PHYSICAL EXAMINATION

The patient appeared apprehensive and depressed, though quick to smile and joke.

Fair state of nutrition; accessory muscles of respiration active at rest. His temperature was 98.6 and respirations 20.

Skin, Scalp, Hair: Non-contributory.

Eyes: Artificial right eye; two-plus arterial changes in left fundus. Pupil round, regular and reacts promptly to light and accommodation.

Ears: Impaired response to whisper on right side.

Nose, Mouth, Tongue, Lips, Gums, Throat: Non-contributory.

Teeth: False.

Neck: No thyroid palpable, trachea in midline.

Thorax: Somewhat barrel-shaped; poor expansion on deep breathing.

Heart: Not percussible; apex not felt; two-plus systolic murmur at apex with normal first sound, though somewhat distant; regular sinus rhythm; pulse 78 and blood pressure 170/90.

Peripheral Vessels: Thickened radials (2-3 plus) ; dorsalis pedis and femoral arteries palpable.

Lungs: Bilateral wheezes and bronchi; numerous sticky rales at each base, persistent after cough.

Abdomen: Symmetrical, non-tender, no masses or herniae.

Genitalia: Normal.

Rectal: Normal; no prostatic enlargement.

Extremities: Slight clubbing of fingers; some wasting of muscles in upper extremities.

Joints: Fusiform swelling of the fingers of both hands with tenderness on compression.

Neurological: Negative.

Fluoroscopic Examination of Chest and Heart: Increased hilar markings; poor diaphragmatic excursions; heart enlarged (2 plus) especially in region of left ventricle; aorta unfolded.

Laboratory Examination: Hemoglobin 15.5 grams; W.B.C. 10,200, 70% polymorphs; urine 1.018, no albumin or sugar, occasional W.B.C. on microscopic examination; Wasserman negative. Electrocardiogram — low Tl, negative T in AVL, high peaked P waves in leads 2 & 3; impression — myocardial changes; P-pulmonale.

CLINICAL DIAGNOSIS

1. Paroxysmal auricular tachycardia.
2. Pulmonary fibrosis and emphysema.
3. Bilateral bronchiectasis.
4. Bronchial asthma, probably intrinsic.
5. Arthritis, both hands.
6. Bursitis, left shoulder.
7. Hypertension; enlarged heart; myocardial fibrosis; regular sinus rhythm.
8. Defective hearing.
9. Artificial eye, right.

CLINICAL COURSE

Because the tachycardia was recurring more frequently and because, except for morphine and a "heroic" dose of ipecac, it had not responded to therapy, he was admitted to the Memorial General Hospital in Las Cruces for observation. There he was seen in several bouts of tachycardia. Bilateral eyeball pressure, unilateral carotid sinus massage, induced gagging, ice bags to precordium and breath holding did not slow the heart. Phenobarbital, one grain five times a day also failed, as did one ounce of ipecac given in divided doses. Quinidine sulphate was then started, grains three (0.2 Gr.) every two hours for four doses without effect. A sudden episode of severe bronchial asthma requiring intravenous aminophyllin and oxygen by mask caused the patient to lose all hope and he left the hospital unimproved on the fourth day.

At home, quinidine sulphate was resumed in higher doses, 6 grains (0.4 Gr.) every two hours by mouth from 8 a. m. to 8 p. m. to a total of 36 grains (2.4 Gr.) per day. On this large dose of quinidine there was no P.A.T. for seventeen days, when a paroxysm appeared. Quinidine was stopped entirely for three days and digitalis then tried. A full average digitalizing dose was given in twenty four hours (1.2 mg. crystodigin) and maintenance ration of 0.4 mg. continued daily thereafter in an attempt to produce nausea as evidence of full or over-full digitalization. The patient did not complain of nausea or loss of appetite and no disturbance of heart rate occurred on this regimen until the thirteenth day, when the paroxysmal auricular tachycardia reappeared.

NO BAD EFFECT

It was felt that since each drug had had some good and no discernible bad effect on the heart a combination of the two might be even more effective. Therefore, without delay, on March 22nd, the patient was told to take 6 grains (0.4 Gr.) of quinidine sulphate every two hours during the waking day and to continue the previous daily ration of crystodigin (0.4 mg.). This joint use of two slightly satisfactory drugs proved to be a

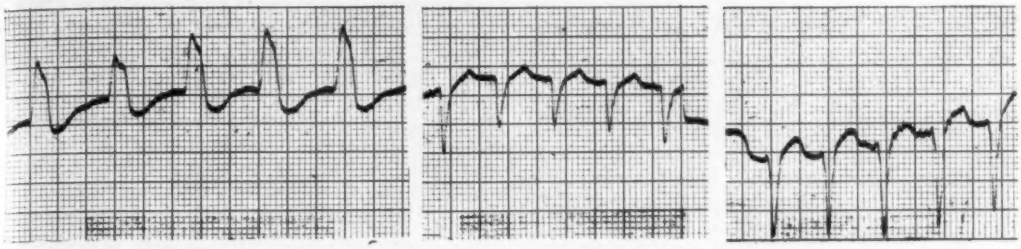

Figure 2. EKG (lead 1) taken 2 hours after one gram of quinidine sulphate by mouth. QRS is 0.16 sec. Quinidine was, of course, stopped at this point.

happy solution, for the patient was free of any significant paroxysmal tachycardia from March 22nd to October. On May 24th and June 12th the patient felt as if his heart was about to "snap" and he was advised by telephone to take three additional grains (0.2 Gr.) of quinidine sulphate immediately and lie down. Each time the threat was stopped and a full-blown tachycardia failed to appear. On May 24th, after taking 0.4 mg. crystodigin for over two months, the patient finally began to lose his sharp appetite and noted a distate for meat, his favorite food. The drug was therefore stopped until his appetite returned (10 days), and was continued thereafter in 0.2 mg. doses. No anorexia or any other toxic symptoms appeared on this smaller dose though it has been used almost continuously for one year.

In July the patient spent three pleasant weeks vacationing with a son in Arizona, remarking that this was the most enjoyable rest he had had in many years. He took quinidine and crystodigin faithfully every day and had no cardiac symptoms whatever.

STARTS ON VISIT

Emboldened by his new-found freedom, in August he started off to visit another son in Denver but on reaching Raton he began to have chills, fever and generalized malaise. He promptly turned back, stopping briefly at Albuquerque for injections of penicillin. At home he was febrile and weak for six days with what was probably a peri-bronchiectatic pneumonitis. His response to antibiotics and oxygen was good. Quinidine and crystodigin were continued as before and no tachycardia occurred. Later in August when he was well over this respiratory infection an electrocardiogram revealed a widened QRS (0.12 sec.) for the first time. In view of the traditional danger attached to this finding he was watched with special care over the next week but nothing extraordinary happened. Since the patient was feeling well and was reluctant to discontinue even one capsule of quinidine the wide QRS was regarded merely expectantly and by the middle of September it had disappeared.

ASTHMATIC DISTRESS

Except for considerable distress from paroxysmal wheezing and coughing due to his asthma (for which he had been using a B.L.B. mask and oxygen for months) nothing of note occurred until October 14, when he was seen at 6 a. m. in advanced pulmonary edema with froth filling his oxygen mask. The patient was hunched in bed, stuporous and sweating profusely; the heart rate was regular at 155 beats per minute, respirations short and rapid. On questioning his family it was learned that the patient had had fairly steady tachycardia for four days but did not report it to us as he felt confident a few extra capsules of quinidine would at any moment stop it. The patient was digitalized rapidly and given aminophyllin and coramine intravenously. A Levine tube was passed and a suspension of 9 grains of quinidine sulphate in tap water inserted into the stomach. Two hours later an electrocardiogram showed no alteration in rate or configuration of QRS and one gram of quinidine sulphate was injected into the tube. Two hours later an electrocardiogram showed the rate to have slowed from 155 to 120, but QRS was now almost 0.16 seconds. Quinidine was therefore withheld. The patient was now responding to questions and his pulmonary edema was improving. Pronestyl was then administered by mouth, three grams being given during the next twenty four hours. With this drug and demerol to allay apprehension and to induce drowsiness, the heart rate fortunately never thereafter rose above 120; during the next 48 hours the heart rate slowed to 90, his pulmonary edema cleared and he made a full recovery. At this point an electrocardiogram showed a normal QRS interval and he was once more given 6 grains quinidine (0.4 Gr.) every two hours but this time for seven doses instead of the previous six (total 2.8

grams from 8 a. m. to 8 p. m.). On this regime of quinidine and 0.2 mg. crystodigin daily he has remained free of tachycardia except for very brief flurrys in November and December. These were controlled by recumbency and an extra 0.2 grams of quinidine sulphate by mouth. The patient's main complaints at the time of this writing (December, 1952, almost two years from the time he was first seen) are dyspnea, cough and wheezing due to severe asthma, bronchiectasis and pulmonary fibrosis.

COMMENT

Three common well recognized points are illustrated by this patient: the danger of addiction to morphine in chronic paroxysmal tachycardia, the appearance of angina pectoris during severe bouts and the liability to congestive failure in older people during prolonged attacks.

Some questions in turn, might be raised. Are quinidine and digitalis safe to use together or do their additive toxic properties make this practice too hazardous? Gold* has stated that their combined use in large doses is to be avoided whenever possible. This is not, however, to be construed as an inescapable dictum, for sufficient evidence has accumulated to indicate their combined use is justifiable in odd cases, especially when the drugs soon prove clearly beneficial and not detrimental. Certainly these two drugs used together have been life-saving in some instances of paroxysmal ventricular tachycardia with congestive failure. Needless to say when the decision is made to use the two drugs simultaneously one must be on guard for evidence of toxicity.

Is it safe to continue quinidine when QRS has widened from .08 to 0.12 seconds? The answer is almost always "no." Gold** presents a useful axiom: " it is not wise to continue the quinidine if the QRS time has been prolonged by about 50 per cent above the control in the particular patient." However, when observation is close, the patient cooperative (or reluctant to decrease the dosage), and the physician fully aware of the dangers, it may be looked upon as a "calculated risk." Thus, on rare occasions it may be regarded as acceptable practice. In this patient the wide QRS became normal while he was taking his usual, rather large doses of quinidine. Again, if a decision is made to continue quinidine despite the widened QRS, close observation is mandatory.

Why did tachycardia occasionally reappear while the patient was taking what was known to be an adequate preventive dose of quinidine? The answer is not certain, but is probable either that the irritable focus in the heart became temporarily more irritable or that the metabolism of quinidine varied from time to time.

*Gold, H. — Quinidine in Disorders of the Heart. Paul Hoeber, Inc., N. Y. 1950, P. 92.

**Gold, H. — idem, P. 68.

CONCLUSION

We report a patient, aged 69, who was plagued by paroxysmal auricular tachycardia for twenty five years, averaging at least one attack a week. He had once become addicted to morphine sulphate which, for years, had been used successfully to stop his attacks. When, finally, he was given digitalis and large doses of quinidine sulphate (2.4 Grams in twelve hours) the patient experienced freedom from significant tachycardia for the first time in two and a half decades. This happy state was abruptly ended, after seven months, by a severe bout of tachycardia lasting four days and ending in severe congestive failure from which he was, fortunately, rescued. Since this episode the dose of quinidine sulphate has been increased to 2.8 Grams in twelve hours and again the patient has been largely free of this disturbing arrhythmia (ten additional months). No special diet was ordered and the patient was permitted to smoke cigarettes since he enjoyed this habit.

New Audio-Visual Aids

New audio-visual aids and uses for those already known are proving extremely valuable in the training of tomorrow's doctors.

Three articles in the July issue of the Journal of MEDICAL EDUCATION emphasize the importance of audio-visual materials in modern medical school teaching.

Devices for illustrating heart conditions to large groups of students are discussed by Dr. J. Scott Butterworth and Dr. Charles A. Poindexter of the Division of Cardiology, Department of Medicine, New York University Post-Graduate School.

A unique device for teaching cardiology is the Educational Electron Cardioscope. The cardioscope was televized from the recent convention of the American Medical Association in Chicago. This instrument is equipped with an audio-amplifier which permits an unlimited number of students to listen to actual heart sounds through individual electronic stethoscopes. At the same time an electronic picture tube traces the electrocardiogram. The instructor may also make the heart sounds visible on the same screen. In addition, the face of the picture tube may be photographed to make permanent reference records of the tracings.

THE DIFFERENTIAL DIAGNOSIS OF CEREBRAL VASCULAR ACCIDENTS*

Cerebral vascular accident, or apoplexy, is now the third leading cause of death in the United States, ranking only after heart disease and cancer.[1] This is readily understandable when it is considered that as the age expectancy of the population increases, more people reach the age at which they are vulnerable to cerebral vascular accidents of various types. Hence, the vascular diseases of the brain assume greater and greater importance, being today the most frequent cause of neurologic symptoms that the physician encounters in his practice. The purpose of this paper is to outline and briefly review some of the conditions concerned in this problem.

In dealing with any given case, three main questions that we must ask ourselves and attempt to answer are:

1. Are we actually dealing with a cerebral vascular accident or has the patient some other condition, general or local, producing signs and symptoms simulating an acute cerebral vascular accident?

2. If the conditions has been established as due to a cerebral vascular accident, what type is it? — that is, are we dealing with hemorrhage, thrombosis, or embolism?

3. What is the site of the lesion? An attempt should be made at localization as this may have some bearing on prognosis. From the standpoint of therapy, however, it is far more important to determine whether the lesion is a hemorrhage, a thrombosis, or an embolism, than to know its location. If it is hemorrhage, a clot is therapeutically desired; if it is thrombosis, treatment is directed toward breaking up and preventing clot formation. Speaking generally, although accurate localization is often an entertaining problem for the neurologist and internist, from a practical point of view the situation is somewhat analogous to a case of myocardial infarction, for even though the cardiologist likes to localize the infarct accurately by means of the electrocardiogram, it makes little difference in the hadling of the patient.

For practical purposes, therefore, the differential diagnosis may conveniently be divided into two parts: (1) differentiation of cerebral vascular accidents from other conditions, and (2) differentiation between intracranial hemorrhage, thrombosis, and embolism.

Prepared by a staff member of the Pratt Diagnostic Clinic, New England Center Hospital.

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DIFFERENTIATION FROM OTHER CONDITIONS

This includes especially those conditions giving rise to acute loss of consciousness.[2] The main ones to be considered here as listed in Table 1, classed according to whether they are extracranial or intracranial, and some remarks about the more important ones follow.

Table 1.—CONDITIONS TO BE DIFFERENTIATED FROM CEREBRAL VASCULAR ACCIDENTS

A. EXTRACRANIAL (diseases not primarily involving the brain)
   1. Toxic states, caused by
      a) exogenous toxins (alcohol, barbiturates, insulin, etc.)
      b) endogenous toxins (diabetic coma, uremic coma, etc.)
   2. Hysterical coma
   3. Adams-Stokes syndrome
   4. Simple fainting
   5. Shock (from injury or hemorrhage)
   6. Heat stroke or freezing (excessive temperatures)

B. INTRACRANIAL (diseases primarily involving the brain)
   1. Postconvulsive coma
   2. Intracranial tumor
   3. Brain abscess
   4. Head injury
      a) concussion
      b) acute epidural hemorrhage
      c) chronic subdural hematoma
   5. Neurosyphilis (G.P.I.)
   6. Encephalitis
   7. Meningitis

In the differentiation between extracranial and intracranial conditions in the comatose patient, it is of extreme importance to look for evidence of unilateral paralysis; if this is present then there is certain to be a cranial or intracranial lesion. It should be emphasized that since deep coma from any cause may result in a bilateral Babinski response, a bilateral extensor plantar response in itself does not indicate an intracranal lesion but may be merely a reflection of the depth of the coma.

BARBITURATE POISONING

Outside of carbon monoxide gas, the barbituric acid derivatives are the most common of the suicidal agents currently in vogue.[3] Any patient that presents the picture of deep coma with flaccidity and complete areflexia presents a possible case of barbiturate overdosage. The pupil of the eye is not diagnostic since it may be normal, dilated, or constricted. Identification of the poison in the stomach contents or urine may be performed.

DIABETIC COMA

Although the differentiation of this condition from a cerebral vascular accident as

a rule presents no serious problem, some confusion may result from failure to realize that glycosuria and hyperglycemia may occur with cerebral vascular accidents in nondiabetic people. This phenomenon is apparently not related to the site of the lesion but is more frequent in patients with cerebral hemorrhage than in those with embolism or thrombosis.[4] Thus, Aring and Merritt[5] in 245 cases of proved apoplexy found 15 patients with glycosuria and moderate to marked hyperglycemia (blood sugar from 160 to 350 mg/100 cc). Only 2 of this group were known diabetics. In addition to hyperglycemia and glycosuria, the presence of diacetic acid in the urine together with a marked reduction in the carbon dioxide combining power of the blood, arbitrarily to 20 vol% or less, is necessary to make the diagnosis of diabetic coma.

ADAMS-STOKES SYNDROME

This syndrome consists in the association of syncope and epileptiform convulsions with marked slowing or temporary cessation of the heart action. The attending coma is necessarily of short duration, not associated with focal neurologic signs, and the electrocardiogram will as a rule demonstrate some degree of auriculoventricular block between attacks.

INTRACRANIAL TUMOR

Hastings[6] aptly stated that "the diagnosis of brain tumor may be a difficult matter at any age. But when tumor occurs at an age which also includes the blood vessel catastrophes, the effects of syphilis on the brain, and the damages of senility, the diagnosis may become very difficult indeed and at times impossible." In spite of these difficulties, however, certain clinical manifestations, observed both in tumor and vascular disease, may with varying emphasis point to the correct answer.[7] The important features to consider are the following.

1. Generally speaking, headaches, is much more severe with tumor, choked disc far more common, and vomiting more distressing. It must be realized, however, that these symptoms may be absent, entirely or in part, in the individual case, especially in the more slowly growing, in the infiltrative, or in the compressing tumor. Further, Riley and Elsberg[8] and others have pointed out that increased intracranial pressure may exist in cases of multiple softening of the brain or cerebral hemorrhage.

2. Although focal seizures may occur at the onset of cerebral hemorrhage, thrombosis, or embolism, repeated focal seizures over a period of time are highly characteristic of tumor and should be considered as due to such a condition until definitely proved otherwise.

3. By far the most important differentiating point is the course taken by each condition, and this can be determined only by a carefully detailed chronologic history or observation over a sufficient period of time. Cerebral vascular disorders of spontaneous occurrence are characterized by suddenness of onset and symptoms and signs seldom progress for more than day or two, after which they remain stationary or improve. This is in contrast to the comparatively gradual onset and steady relentless progression of signs and symptoms inherent in the development of intracranial tumor. Although the progressive course is usually a slow one, hemorrhage into a tumor may of course cause rapid progression with rapid loss of consciousness.

ACUTE EPIDURAL HEMORRHAGE

This is due to rupture of the middle meningeal artery after a head injury. The unconsciousness due to concussion may pass without interruption into that due to hemorrhage or there may be a lucid interval in which consciousness is recovered and the patient is apparently normal. This interval may last several hours before unconsciousness redevelops and sedatives should therefore never be given as they may mask this lucid interval. Epidural hemorrhage leads to progressively deepening coma with signs of a focal lesion of one hemisphere often beginning with convulsions and leading to hemiplegia.

CHRONIC SUBDURAL HEMATOMA

This is a late result of head injury and may develop weeks and even months after an accident. The head injury may be so trivial as not even to be remembered. A "cystlike tumor" forms in the subdural space and gradually expands with the passage of time. This results in a steadily progressive course closely resembling that of intracranial tumor and characterized by increasing headache, drowsiness, confusion, and ultimately coma. Because it does not indent the underlying cerebral hemisphere as an epidural hemorrhage does, but tends to push the whole brain aside, signs of focal cerebral compression are frequently absent. The definite exclusion of chronic subdural hematoma may be very difficult at times; in doubtful cases burr holes should be made as this condition will ultimately lead to death if left untreated.

DIFFERENTIATION BETWEEN VARIOUS TYPES OF APOPLEXY

Having considered the differentiation of cerebral vascular accidents from other con-

ditions, we now come to the second phase, namely, the differentiation between the various types of cerebral vascular accidents. These have been grouped together as indicated in Table 2.

Table 2.—TYPES OF CEREBRAL VASCULAR ACCIDENTS

A. INTRACRANIAL HEMORRHAGE
   1. Intracerebral
   2. Extracerebral
      a) traumatic (acute epidural hemorrhage; a) chronic subdural hematoma)
      b) spontaneous subarachnoid hemorrhage
B. CEREBRAL THROMBOSIS
C. CEREBRAL EMBOLISM
D. HYPERTENSIVE ENCEPHALOPATHY

SPONTANEOUS CEREBRAL HEMORRHAGE AND CEREBRAL TREATMENT

These conditions are considered together because they have many clinical similarities and yet their differentiation in a given case is important from the standpoint of prognosis and therapy. The distinction between hemorrhage and thrombosis in a given case may be extremely difficult and "often appears more impressive on paper than during anxious moments at the bedside."[9] Although no one clinical sign will differentiate the two condtions with absolute accuracy, the various points indicated in Table 3 help in discriminating between hemorrhage and thrombosis.

Table 3.—HEMORRHAGE AND THROMBOSIS

A. CEREBRAL HEMORRHAGE
   No prior seizures
   Onset with headache and vomiting and maybe convulsion
   Progressive unconsciousness
   CSF pressure greater than 300 mm H_2O
   Bloody CSF
   Stiffness of neck
   Bilateral Babinski
   Eye abnormalities

B. CEREBRAL THROMBOSIS
   Prior seizures
   May be convulsions at onset
   Short initial phase of unconsciousness
   CSF pressure less than 300 mm H_2O
   May be microscopic blood in CSF only
   Monoplegia
   Hemianopia
   Persistent aphasia

It might be pointed out that the symptoms occurring at the onset are of some aid. Premonitory symptoms are just as apt to occur with hemorrhage as with thrombosis but the occurrence of a sudden severe headache and vomiting at the onset is strongly indicative of hemorrhage. Progressive deepening unconsciousness is highly suggestive of bleeding.

The findings at lumbar puncture are of greatest aid. Aring and Merritt[5] found that the cerebrospinal fluid pressure was greater than 300 mm H_2O in 38 per cent of the cases of cerebral hemorrhage and greater than 400 mm in 18 per cent. In the cases of cerebral thrombosis, a pressure greater than 300 mm was rare and a pressure greater than 400 mm did not occur. A frankly bloody fluid is indicative of cerebral hemorrhage but occurs in only about 75 per cent of these cases. Microscopic blood and slight xanthochromia may occur in cases of cerebral infarction due to thrombosis. The mortality rate in cerebral hemorrhage is much higher than in cerebral thrombosis, a cerebral hemorrhage of any appreciable size being invariably fatal.

CEREBRAL EMBOLISM

The incidence of cerebral embolism may be higher than ordinarily realized. In an analysis of 329 autopsied cases of vascular diseases of the brain, Adams[4] placed the incidence of embolism in the neighborhood of from 20 to 30 per cent. The outstanding clinical feature of cerebral embolism is the suddenness of onset, so that the immediate disturbance of brain function is maximal at the onset. The course is usually one of improvement, at least temporarily. Clinically, the diagnosis can be made only when there is an obvious source or evidence of multiple emboli to various organs. The usual source of a brain embolus is in the left heart, the common etiologic conditions being auricular fibrillation, endocarditis with vegetation formation. myocardial infarction with a mural thrombus, and mitral stenosis. With these considerations, the diagnosis of brain embolism is usually not a difficult one.

SPONTANEOUS SUBARACHNOID HEMORRHAGE

This is invariably due to a ruptured saccular aneurysm at the circle of Willis at the base of the brain. Individual cases rarely vary from the classical syndrome, whose diagnostic features are:

1. Sudden onset—frequently accompanying muscular exertion.
2. Severe headache—usually occipital and in the back of the neck.
3. Signs of meningeal irritation — stiff neck, Kernig's sign.
4. Absence of lateralizing signs—a bilateral Babinski may be present in a comatose patient.
5. Bloody cerebrospinal fluid within the first 24 hours, followed by xanthochromic cerebrospinal fluid for 2 weeks. In contrast to intracerebral hemorrhage, coma or convulsion at the onset is rare.

HYPERTENSIVE ENCEPHALOPATHY

Pickering[10] and others have pointed out that hypertensive encephalopathy comprises

at least two distinct clinical and pathologic conditions. One type is associated with an acute rise in blood pressure and is characterized by headache, vomiting, bradycardia, convulsions, and coma. These symptoms are due to acute edema of the brain. The second type is more common in cases of chronic hypertension, and focal signs and symptoms dominate the picture. Clinically, the most striking feature is sudden attacks of sensory or motor paralysis, usually to a very limited extent, of brief duration, and with a tendency to complete resolution — for example, transitory aphasia, monoplegia, or hemianopia. Consciousness is usually retained. These episodes are felt to be due to focal ischemia of the brain, either secondary to local vasospasm or to actual organic arterial obstruction.

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Complaint Circa 1872

"Forty years ago this spring a leading member of the faculty of the Harvard Medical School in an address on medical education asserted that there was grave danger of devoting too much time to science in the medical course. He was fearful that as a result the schools would turn out scientists instead of practical doctors. Not only was there open hostility to science, but a hearty endorsement of the plain rule of thumb practice in medicine and surgery. 'The student's work', he said, 'is mainly with facts of empirical association.'

"There are doubtless some teachers still living who would endorse the sentiment of Trousseau, the distinguished French physician, who said in his clinical lectures, 'For mercy's sake, gentlemen, let us have a little less science and a little more art.'

"Helmholtz states that within his memory vitalistic views prevailed to such an extent that many physicians in Germany objected to the use of auscultation and percussion because such gross methods of examination implied that man was a machine rather than a living spirit." — Pratt, J. H., "The Method of Science in Clinical Training," *Boston M. & S. J.* 166:835, 1912.

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